A CLINICAL STUDY OF POISONING IN BUNDELKHAND REGION,

THESIS

For

DOCTOR OF MEDICINE

(MEDICINE)

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C E R T I F I C A T E

This is to certify that the work entitled "A CLINICAL STUDY OF POISONING IN BUNDELKHAND REGION" which is being submitted as a thesis for M.D.(Medicine) Examination, 1994 of Bundelkhand University, has been carried out by Dr. Vinod Kumar Verma under my direct supervision and guidance.

The techniques embodied in this work were undertaken by the candidate himself. The results and observations were checked and verified by me from time to time.

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Certified that the work entitled "A CLINICAL STUDY OF POISONING IN BUNDELKHAND REGION" which is being submitted as a thesis for M.D.(Medicine) Examination, 1994 of Bundelkhand University, has been carried out by Dr. Vinod Kumar Verma in the department of Medicine, M.L.B. Medical College, Jhansi.

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C O N T E N T S

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I N T R O D U C T I O N

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I N T R O D U C T I O N

A poison is a substance (solid, liquid or gaseous) which if introduced in living body, will produce ill health or death by its constitutional or local effect or both (Reddy, 1982).

There is really no demarkating level between a medicine and poison, for a medicine in a toxic dose is a poison and a poison in small dose may be a medicine. In law, the real difference between a medicine and a poison is the intent with which it is given. If the substance is given with the intention to save life, it is medicine, but if it is given with the intention to cause bodily harm, it is poison (Parikh, 1985).

Poisoning may be suicidal, homicidal, stupefying or accidental. All types of cases of poisoning are comparatively more common in India than in advanced countries because of ease with which poisonous chemical are available, the carelessness with which they are stored. It is a sad fact that cases of poisoning of all types are increasing day by day. Accidental poisoning commonly takes place as a result of carelessness with which the poisonous and nonpoisonous materials are stored together.

Accidental poisoning is common in childhood. Children in the preschool age are particularly vulnerable

due to their intense desire to experiment with an explore their environment. The present day households lurking with toxic substances at every corner, such as caustics, insecticides and medicines, provides an all to easy setting for susinquisitiveness to end in disaster (Srivastava et al, 1990).

Poisoning may be local (limited to eye, skin etc) systemic or both depending on dose, extent of absorption and distribution, intrinsic potency, and host susceptibility. Absorption and distribution are influenced by properties of chemical itself and of biological barrier through which it penetrates, local effects are due to non specific chemical reaction. Such as oxydation protein denaturation (Frederick et al, 1990).

CLASSIFICATION OF POISON

No classification of poisons is entirely satisfactory, as many poisons fall into more than one group. However, classification given below according to the mode of action of poison, is the one most commonly used. Accordingly poisons are classified in six groups (Naik, 1986).

I. CORROSIVES

A corrosive poison is simply highly active irritant and not only produce inflammation but also actual ulceration of tissues. They are :-

a. Strong acids : Sulphuric acid, nitric acid, hydrochloric acid.

b. Strong Alkalies : Caustic soda, caustic potash.

II. IRRITANTS

Irritant poisons produce symptoms of pain in the abdomen, vomiting and purging.

a. Inorganic : Arsenic, lead, copper, phosphorus, iodine etc.

b. Organic : Caster oil seeds, abrus precatorius etc.

c. Mechanical : Dried sponge, powdered glass etc.

III. NEUROTICS

Neurotics poisons act chiefly on nervous system though some neurotics have a local irritant action. They are :

a. Cerebral

i) Somniferous - opium.

ii) Inebriant - Alcohol, ether, chloroform, sedatives and hypnotics, fuels (petroleum, kerosene) and insecticides.

b. Spinal : Nux vomica.

c. Peripheral poisons : Curare, conium.

IV. CARDIAC : Digitalis, oleander, tobacco etc.

V. ASPHYXIANTS : Coal gas, sewargas, methyl isocynide gas (Bhopal gas Tragedy), CO, CO_2, phosgene gas etc.

VI. MISCELLANEOUS

As name suggests poisons having different action are put together in this groups viz. analgesic and anti-pyretics, tranquilizers, antidepressants, stimulants etc.

Increasing incidence of pesticide poisoning is due to increasing uses of pesticides in agriculture and public health for vector control. Pesticides is a poison use to destroy pest of any sort (Park et al, 1986). The word pesticide is a general term that includes :

1. Insecticides :
   a. Organochlorine compounds : DDT, Elderin, B.H.C. etc.
   b. Organophosphorus compounds : Malathion, parathion, diazenon etc.
   c. Carbamates compounds : Carbaryl, propoxus (OMS 33) (Baygon).
2. Rodenticides : Aluminium phosphide and zinc phosphide etc.
3. Fumigants : Poisons substances contact with water or moisture, release gas, e.g. zinc phosphides, aluminium phosphide.

FACTORS MODIFYING THE ACTION OF POISONS

The factors which modify the action of poisons are dose, form of poison, method of administration and condition of body. As a general rule, the deleterious effect of a poison depend on its dose. If the quantity administered is small, there may be less toxic effects;

if the quantity administered is large, severe symptoms usually follows quickly resulting in serious toxic effects including death. Gaseous or vapours acts more quickly than fluid poisons because they are absorbed immediately. liquid poisons act more quickly than solid ones of which fine powders acts more quickly than course ones. The toxic effects of substances may vary greatly according to their solubility or insolubility resulting from a chemical combination, action of poison is considerably altered when combined mechanically with inert substances for e.g. when alkaloids taken with animal charcoal fail to act. Poisons act slowly when the stomach is full with fats.

The rapidity of action of poison also depends upon the mode in which it is introduced into the system. Thus a poison acts most rapidly when inhaled in a gaseous or vapours form or introduced into blood current by injection in vein, by subcutaneous or intramuscular injection (Modi, 1985; Lawson, 1986 and Parikh, 1986).

MANAGEMENT OF POISONING

In practice most emergency treatment of acute poisoning is symptomatic and not specific to poisons. Success depends upon combination of speed and common senses as well as on the poison, the amount taken, and time which has since elapsed. In clinical practice a selective antidote is available in less than 2% of episodes (Laurence et al, 1980).

1. Identification of poison : because some poisons have selective antagonist.
2. Removal of poisons by vomiting and gastric lavage. Generally it is advocated that gastric aspiration should be done within 4 hours of ingestion of drug, but upto 12 hours or longer with ingestion of salicylate or any drugs with anticholinergic action which remains longer in stomach. Emesis and lavage are contraindicated in corrosive poisons but decision can be influenced by amount taken and its concentration.
3. Prevention of further absorption of poison by specific antidote and universal antidote.
4. Forced diuresis.
5. Supportive management :
   a. Airway
   b. Maintain blood pressure.
   c. Care of back bladder and bowel.
   d. Nutrition.
6. Haemodialysis.

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REVIEW OF LITERATURE

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REVIEW OF LITERATURE

It has been a common observation that the incidence of poisoning has increased in last few years. This is attributed to rapid development of chemistry, leading to availability of newer compounds in trades, industries and medicine. Easy access to these substances, increasing stress, strain in modern time predispose to increased risk of suicidal poisoning, whereas accidental exposure occurs in industrial settings and in children consuming them out of curiosity. Homicidal poisoning is seen when revenge or personal gain is the factor. Organophosphorus and organochloride insecticides, rodenticide, ethyl alcohol, hypnotics and sedatives constitute majority of cases of poisoning. Region wise, organophosphorus poisoning is common in Maharasthra and Tamilnadu. Rodenticide (Rat poison) and copper sulphate poisoning is common in Uttar Pradesh and Madhya Pradesh (Anjaria, 1988).

On the basis of annual reports of chemical examination the common pattern of poison in state of Uttar Pradesh and Madhya Pradesh in the year of 1972, 1973 and 1974 is given table below (Tiwari, 1975).

Name of poison	Percentage of total poisoning cases		
	1072	1973	1974
Alcohol	28	21	30
Insecticide	25	16	26
Zinc phosphide(Rodenticide)	21	20	14
Dhatura	9	10	7
Sedatives (Barbiturate, Morphine, Psychotrophic drugs)	10	7	9
Copper sulphate	2	3	2
Others (Methylene, Methyl alcohol, cynide)	5	23	12

The pattern of poisoning in state of Bihar in 1968, 1969 and 1970 is given below (Modi, 1985).

Name of poison	Percentage of poisoning		
	1968	1969	1970
Organophosphorus	40	68	72
Endrin	32	6	7
Zinc phosphide (Rodenticide)	13	9	11
Dhatura	8	10	4
Alcohol	3	3	1
Miscellaneous	4	4	5

According to Pohwala and Ghai (1956) accidental poisoning in children is a global problem. Accidental

poisoning is a 12th leading cause of admission in paediatric wards in India and account for about 1% of hospitalised patients. Most of cases of accidental poisoning were preventable. The pattern of accidental poisoning in children in central India is shown in table below.

Accidental poisoning	Percentage
Kerosene oil	30.2
Poison plants and seeds (Dhatura)	27.2
Household medicine (Aspirin etc.)	23.5
Insecticide, DDT, Naphthalene	8.1
Food poisoning	6.6
Miscellaneous	4.4

In 1972, on the basis of theoretical model a WHO expert committee made the 1st global estimate for the number of cases of acute pesticide poisoning. This indicated that problem was large and required urgent attention. Data supporting the model were subsequently obtained through the study in Indonesia, Malasiya, Srilanka and Thailand. It is largely a problem of developing countries.

The extent of problem of acute pesticide poisoning has been highlighted as a major health concern in the Srilankan study. It was noted that in the year of study, 982 deaths from acute pesticide poisoning in State hospitals were reported. This figure strongly demonstrate public

health importance of the problem, as it was almost twice as high as the total number of deaths in same hospital because of Malaria, tetanus, the traditional killers in the developing countries. The study in developing country found that approximately 2/3rds of all acute pesticide poisoning were suicidal attempts and approximately 1/4th were accidental. In remaining cases, the cause was not defined. Pesticides are often used to commit suicide in these countries because these hazardous compounds are readily available to public (Jayaratnam, 1982).

Acute poisoning is an important medical emergency accounting for a significant proportion of admissions in the medical emergency wards. Three hundred sixty patients with acute poisoning comprising 2.15% of all medical admission in a year were analysed by Kumar et al (1988). Males out numbered females (283 : 77), age range was 13-70 years (mean age 28.7 years). Almost three fourths (73.3%) cases were in the 2nd and 3rd decades of life. Dhatura poisoning was the commonest (78, 21.66%) followed by alcohol poisoning (42, 11.66%), CNS depressant viz. sedative and psychotrophics (28), Barbiturate -9, dilantin-1 and opium - 2 (Total 40 cases, 11.11%), copper sulphate (26 cases, 7.22%), corrosive (16 cases, 4.4%), aluminium phosphide and rodenticide poisoning were seen in 2.5% and 2.7% cases respectively. Two cases of dog killer poisoning were also encountered, miscellaneous group (20 cases, 5.5%)

including poisoning by diverse substances viz. INH, Dapsone, Hg, potassium dichromate, carbon mono-oxide, ether, bhang and common household chemicals. Dettol, tincture iodine, camphor, phitkari, neel, naphthalene etc. exact poisons could not be identified in 66(18.33%) cases. The intention was suicidal in 177 (49.16%), accidental in 29 (8.05%) and homicidal in 6 (1.65%) cases. It was interesting that in 73 (20.27%) cases poison was used to stupify to rob the patients, 21 patients died and mortality rate was 5.8%.

Samaria et al (1988) carried out a study of poisoning in eastern UP and Western Bihar. They analysed 225 patients of different poisonings attended the casuality department during a period of one year in Institution of Medical Sciences, Varanasi. Out of total 225 cases, 175 belonged to UP and 49 belonged to Bihar and one patients was from M.P. Of the total 120 (53.4%) were females and 105 (46.6%) cases were males. Maximum cases belonged to age group (11-20) years followed by age group 21-30 years (23.12%) and the age of 31-40 years (10.66%). Much less number of cases belonged to the age group of ⩽10 years and ⩾40 years. Majority of the cases (160, 71.2%) were suffering from organophosphorus poisoning followed by 20 (8.88%) cases of diazepalm poisoning, 15(4.45%) of Barbiturate poisoning, 7(3.12%) cases of petroleum product like kerosene oil, petrol etc., 3(1.34%) of corossive poisoning i.e. by acid and alkali and salicylate

phenothiazine poisoning was less common having only one patients each (0.44%).

From a decade, insecticides like aluminium phosphide and endosulphan are being frequently used for poisoning. Maltani et al (1989) conducted a study of changing spectrum of acute poisoning in 106 adults in period of 6 months in Medical College, Hospital, Patiala. Organophosphorus compound was the commonest agent(44.5%), aluminium phosphide (20%), endosulphan (16.36%), alcohol (7.27%), kerosene oil (2.3%), copper sulphate (1.82%), opium (1.82%), diazepalm (0.91%) and mixed (3.64%). Ninety four (85.45%) cases were males and sixteen(14.55%) were females and peak age was between 21-30 years(43.18%). Acute poisoning was more common in lower income groups (56.36%) and in rural areas (64.54%). In addition to information supplied by the patients and/or attendants, clinical examinations helped in making the diagnosis. The mean hospital stay was 2-7 days and majority of patients stayed less than 24 hours (58.18%), over all mortality rate in this study was 25.5%. It was highest with aluminium phosphide poisoning (77.2%), followed by mixed poisoning and organophosphorus poisoning (25% and 18.32% respectively). Easy availability and quick lethal action of aluminium phosphide and endosulphan has prompted frustrated people to consume these agents, lack of awareness, regarding methods of spraying caused organophosphorus

compound poisoning in large number of cases.

The clinical examination from 121 patients of acute poisoning admitted through casuality department in Medical College, Hospital, Cuttuck within the period of 2 years, were analysed by Mahapatra et al (1991). They showed that there were 77 (63.6%) males and 44 (36.3%) females patients. The mean age was 27.2±10.4 years and it was commonest (40.49%) in age group of 21-30 years followed by 11-20 years (25.6%) and then progressively declining over the years. Organophosphorus compound poisoning was the commonest (28.9%) followed by Oleander (17.3%) and adultered alcohol (17.3%) intoxication. There was self induced poisoning in 90(74.3%) cases and accidental in 31(25.6%) patients.

Chitalkar et al (1991) conducted a retrospective study of forty two patients with poisoning admitted to Military Hospital, Ambala Cantt during an eighteen months period. Their age ranged from eighteen months to forty eight years with mean age of 21.6 years. Twenty were males and 22 were females. They took a total of eighteen different substances. Organophosphorus in eight (18.9%) and aluminium phosphide in 6(14.2%) were the commonest, 18 patients consumed poison with suicidal intent and ten consumed it accidentally. The cause remained unknown in twelve. Two cases were given dhatura homicidally.

Psychiatric evaluation showed at least six individuals with depression. Four of forty two (9.1%) died.

Clinical profile of 100 cases of poisoning admitted in acute medical care unit were studied in Vishakhapatnam from January, 1992 to 31st May, 1992. Fifty five percent cases were males and 45% cases were females. Majority of cases (74%) were under the age of 30 years. No case of poisoning was recorded after the age of 50 years. The commonest type of poisoning in this study was due to organophosphorus and allied compounds (35%), followed by drugs (22%), ant poison (Gamanaxene-20%), rat poison (zinc phosphide - 10%), alcohol - 5%, Nerium seeds - 4%, phenol - 3%, sulphuric acid - 1%, (Pandurangarao and Krishna Murthy, 1992).

ALUMINIUM PHOSPHIDE POISONING

Aluminium phosphide is widely used for grains and fruits preservations against rodents and insects (Roy and Bedi, 1979). Aluminium phosphide is a solid fumigate pesticide. It is marketed in India as a tablet of celphos and quickphos manufactured by Oxcel Laboratory and Inventa Corporation respectively. It was declared as an ideal pesticide because of its being cheap, most efficatious and easy to use (Thomas, 1973). Its effects were due to liberation of phosphine gas which is toxic to pests, insects and rodents. After fumigation the non toxic residue left in the grains are the

phosphite and hypophosphite of aluminium.

Human toxicity which is usually acute, occurs either due to inhalation of phosphine from fumigated grains (Wilson et al, 1980) or after ingestion of aluminium phosphide. The latter liberates phosphine in the stomach and this is widely absorbed through out the gastrointestinal tracts. Earlier acute intoxication was limited to inhalation of phosphine, but in last decade poisoning due to aluminium phosphide ingestion has been reported from different northern states (Jain et al, 1985; Agrawal et al, 1989; Khosla et al, 1990).

This acute ingestional toxicity has become a common cause of death among young generation in northern India. In India the first case was reported in 1981, during 1984-85 study of few cases appears in literature on its epidemiological and electrocardiographic aspects. It prevalence rate calculated on the basis of hospital admission was 2-3 cases per thousand admissions and its surpassed every other poisoning in Haryana. The poisoning is more common in young adults, mostly in their teens. The mode of poisoning is usually intentional, occasionally accidental and rarely homicidal. The agricultural community irrespective of sex is more at risk due to illiteracy and easy availability of pesticide in household. The peak season is from May to September.

Organ damage is wide spread, its pathogenesis is still not clear (Khosla et al, 1990). The damage is hypoxic in nature and due to binding of cytochrome oxidase at cellular level. The acute cardiotoxicity is also due to subcellular transmembrane exchange of ions $(Na, K, Mg \text{ and } Ca)^{+}$ brought on by focal myocardial necrosis produced by phosphine. The high mortality is due to rapid onset of symptoms delay in arrival of hospital, delay in instituting treatment, lack of an antidote, non responsiveness of shock to resuscitative measure and consumption of large dose (Chug et al, 1989). Little attention has been paid to literature to management of these cases and even standard text book do not deal with the subject in details despite increasing awareness of this poisoning.

Each tablet of celphos or Quick phos (3.0 gm) contains 56% aluminium phosphide and 44% ammonium carbonate and had the capacity to liberate 1.0 gm of phosphine gas. The lethal dose for human is 150-500mg for a 70 kg person. (Sidney, 1980). The gases liberated during its reaction with water, humidity are phosphine and ammonia etc.

Aluminium phosphide + 3 H_2O -- $Al(OH)_3$ + PH_3 (Phosphine)

$(NH_4)_2\ CO_3 + H_2O$ --- $2NH_4OH + CO_2$ -- $NH_3 + CO_2 + H_2O$
(Ammonia)

CLINICAL FEATURES

The clinical symptomatology is more or less same irrespective of the mode of toxicity, except that the initial symptoms pertain to the route of entry. The signs and symptoms depend on the dose and severity of poisoning. In mild ingestional intoxication severe systemic features do not occur except G.I.T. symptoms viz. nausea, vomiting, headache and abdominal pain or discomfort, and these patients usually recover. On the other hand systemic manifestations are early and progressive in moderate and severe poisoning and most of times prove fatal. Initial gastrointestinal symptoms may be followed by shock (Singh et al, 1985 and Chopra et al, 1986). Chug et al (1991) reported the following symptoms and signs of moderate to severe aluminium phosphide poisoning :-

a. Gastrointestinal symptoms are nausea, vomiting, diarrhoea and retrosternal pain.

b. Cardiovascular - hypotension or shock, changes in heart rate (bradycardia or tachycardia), myocarditis, pericarditis, acute congestive heart failure and ECG changes.

c. Respiratory - Cough, dyspnoea, cyanosis, rales and rhonchi, respiratory failure (Type-I).

d. Hepatobiliary - Jaundice, tender hepatomegaly, raised transaminases, enzymes.

e. Renal - oliguric and non oliguric renal failure.

f. Central Nervous System - Headache, dizziness, restlessness without alteration of consciousness, acute hypoxic encephalopathy.

The clinical paramter of aluminium phosphide poisoning were studied by various authors in different states. The incidence of aluminium phosphide poisoning in Haryana was unknown before 1980 but now its incidence is progressively increasing not only in Haryana but in the whole of Northern India. The incidence of aluminium phosphide poisoning was 0.06 per thousand of hospital admissions in 1981 and steadily increased to 10 per thousand in 1989 in Medical College Hospital Rohtak (Chug et al, 1991). Majority of patients (92%) were belonging to age group of 15-40 years. The male : female ratio was 40 to 73% patients and male 27 to 60% females. Sixty to 85% patients were belonging to rural area and in 71 to 97% of cases the poisoning was intensional (suicidal) in nature Sepaha et al, 1985; Singh et al, 1985; Siwatch et al, 1988; Agarwal , 1989; Chug et al, 1991; Khaniju et al, 1990; and Khosla et al, 1992).

The clinical spectrum of aluminium phosphide poisoning gastrointestinal upsets such as nausea, vomiting, shock (61-90%), tachycardia (51-74%) patients, 30 to 50% patients had S_3 and S_4 gallop, tachypnoea, dyspnoea, crepts and rhonchi, 20-30% patients had altered sensorium, 8-10% had acute renal failure, 1-5% patients had tender

hepatomegaly and 3% patients had bradycardia (Katira et al, 1990; Khosla et al, 1990 and Chug, Dushyant and Santram, 1992).

In aluminium phosphide poisoning ECG changes were present in 75-95% of cases. They were ST-T changes (both ST elevation and depression) in 30-65%, atrial fibrillation in 50 to 60%, sinus tachycardia 20 to 40%, right bundel branch block 15 to 30%, sinus bradycardia 10 to 15%, ventricular ectopics 10 to 15%, T wave inversion 10 to 14%, cases, left bundle branch block 10%, idioventricular rhythm 10%, wandering pacemaker 3 to 5%, ventricular tachycardia 5%, tachybradycardia syndrome 5% (Trivedi et al, 1982; Jain et al, 1985; Raman and Dubey, 1985; Chopra et al, 1986; Chug et al, 1989 and Katira et al, 1989).

Aluminium phosphide poisoning is a life threatening emergency with a high mortality and no specific antidote (Khosla et al, 1992). Mortality has been highly variable (37-100%)(Chopra et al, 1986; Ram et al, 1988 and Bajaj et al, 1989). Mortality depend on the dose of poison, freshness of the compound (Katira et al, 1990), duration and severity of shock, presence or absence of bad prognostic factors, and complications. The bad prognostic indices included poor response of shock to dopamine infusion, anaemia, chest infections, metabolic acidosis, severe hypoxia, electrolyte disturbances, presence of arrhythmia and aspiration, pneumonia (Chug et al, 1990).

The complications included pericarditis, acute congestive cardiac failure, acute massive gastrointestinal bleeding and respiratory failure (Chug et al, 1991). The time interval between ingestion and death varied from 4 to 40 hours with a mean of 12.1 hour. The commonest cause of death was peripheral circulatory failure (76%), remaining died due to recurrent ventricular fibrillation (9%), acute pulmonary oedema (9%) and fulminant hepatic failure (6%), (Agarwal, Agarwal and Jain, 1989).

ORGANOPHOSPHORUS POISONING

Organophosphorus insecticides are chemical agents that have a powerful inhibitory action on enzyme acetylcholinesterase. This is due to the fact that their phosphate radicals firmly bind to action site of enzyme to form stable phosphorylated complexes. These substances thus acts as anticholinesterase agents and results in accumulation of acetylcholine at cholinergic nerve endings throughout the body. The overabundance of acetylcholine initially stimulates and then paralysis impulse transmission in cholinergic synapses. Cholinergic transmission occurs in widespread areas of the body including autonomic ganglia, parasympathetic nerve endings, somatic nerves, cells of the central nervous system and some special sympathetic nerve endings such as in sweet glands. It is this wide spread distribution of cholinergic nerve endings

in the body that accounts for diversity of clinical features in organophosphorus poisoning (Gupta et al, 1988).

Pharmacological organophosphorus compounds have three types of actions (a) Muscaranic (b) Nicotinic and (c) on the central nervous system.

As a result of the muscaranic like effect the following signs and symptoms are observed.

A. Bronchial Tree : Tightness of the chest with prolonged wheezing expiration suggestive of bronchoconstriction and increased secretion. Therefore there is discomfort or pain in chest, dyspnoea, cough, pulmonary oedema and cyanosis. The effect stimulates bronchial asthma.

B. Gastrointestinal : Anorexia, nausea, vomiting, abdominal cramps, epigastric and substernal tightness with heart burn and eructations, diarrhoea, tinismus and involuntary defaecation.

C. Increased sweating , salivation, lactimation, tears, may be red due to porphyria in lacrimal glands.

D. Heart bradycardia.

E. Pupils - Slight miosis, occasionally unequal and later more marked miosis.

F. Urinary bladder frequency of micturition and involuntary micturition.

As a result of nicotine like effects, the following signs and symptoms are observed. Straight

muscle - easy fatigue, mild weakness, muscular twitching, cramps, fasciculations, generalise weakness of muscles' of respiration with diarrhoea and cyanosis.

As a result of action of central nervous system the following effects may appear approximately in the order mentioned below :

1. Irritability, apprehension, restlessness.
2. Fine fibrillary tremors of hands, lids, face and tongue.
3. Mental confusion progressing to stupor and muscular weakness with tremors and convulsions.
4. Coma with absence of reflexes and depression of respiratory and circulatory centre (Parikh, 1986).

Incidence of suicidal attempts with consumption of organophosphorus compound is increasing in recent years because of frustration and unemployment (Bitchili et al, 1990)

The clinical spectrum of organophosphorus poisoning were studied by various authors in different states. There were 60-70% of male cases and 30-40% cases were females. Majority of patients (80%) belonged to rural area (Bhakat ram et al, 1989; Ghosh et al, 1991).

Clinical finding of organophosphorus poisoning were nausea, and vomiting (80-100%), altered sensorium (60%), constricted pupil (60%), pulmonary oedema (60%), excessive sweating, frothings, lacrimation, salivation

(30-60%), abdominal pain (30%), abnormal behaviour, ghabrahat, restlessness, giddiness (10-15%) and pulse rate varied from 32 to 140/min (Vishwanathan et al, 1962; Gupta et al, 1968; Surjeet et al, 1969; Gupta et al, 1989). The over all mortality was found to be 15-45% (Bhakt ram, et al, 1989; Chamunderswari, 1990; Ghosh et al, 1991).

Sennanayke and Kanaliedda (1987) described yet another manifestation of neurotoxicity following the cholinergic illness of organophosphorus compounds, a paralytic syndrome was observed in 5-10% of patients with organophosphorus poisoning. Muscles weakness developed 24 to 96 hours after the cholinergic illness, involving primary the proximal limb muscles, neck flexors, certain cranial motor nerves and the muscles of respiration, because of weakness of muscles of respiration. Seventy percent patients had difficulty in breathing and 30% patients died from respiratory failure. This condition called intermediate syndrome and was not responsible for atropine and prolidoxime and required urgent respiratory support.

Bitchili et al (1990) reported interesting neurological deficit besides muscarnic and nicotinic manifestations of organophosphorus poisoning. They were pancerebellar syndrome, proximal muscle weakness and sluggish reflexes, neck muscles weakness, ptosis.

Twenty five to forty five percent of cases of organophosphorus poisoning had some ECG changes. ECG changes showed sinus bradycardia (10-20%), sinus tachycardia (15%), proximal atrial tachycardia (10%) and 5% cases had ventricular premature beats (Singh and Malhotra, 1991 and Bitchili et al, 1990).

ZINC PHOSPHIDE (RODENTICIDE POISONING)

Zinc phosphide is an efficient rodenticide when moist the chemical slowly gives phosphine, whose garliac odour is repellent to man and domestic animals. But seems to have no adverse effects on rats. Zinc phosphide now extensively used in India. It is used in the ratio of 1 part to 10 part of wheat or rice flour and mixed with a few drops of edible oil in order to render it more attractive to rats. Because of its good safety records, low cost and reasonably high effectiveness, zinc phosphide is recommended for large scale use against rat (Park and Park, 1986).

Commercial zinc phosphide is one of the well known rat poisons, it is a mixture of different phosphides of zinc in addition to traces of zinc phosphate and zinc oxide. The chief symptoms after the administration of zinc phosphide are vacant look, frequent vomiting with retching, tremors and drowsiness followed by respiratory distress and death. Zinc phosphide acts in the stomach

with liberation of phosphine which acts as a respiratory poison (Modi, 1985).

In 1989 Subramanium carried out a study of 712 cases of poisoning admitted in toxicology department in Madras during 2 years. Out of which 32 cases were of zinc phosphide (rat killer) poisoning. The death rate of rat killer poisoning was 6% when compared to north Indian States (25%).

DHATURA POISONING

Dhatura plant is known as thorn apple and commonly grows in waste places all over India. The fruits are spherical and have sharp spines giving the name thorn apple to plant. An average sized fruit contains 450 to 500 seeds and weight about 8 gms. Dhatura is poisonous in all its parts but the seeds and fruits are considered to be most noxious. The active substance is known as dhaturine and contains the alkaloids leavohyoscyamine, hyoscine or scopolamine and traces of atropine. The alkaloids of dhatura powerfully stimulate the higher and other centres of the brain and also exercise at same time a paralysing action on various nerves. They inhibit secretion of sweat and saliva, dilate the cutaneous blood vessels and pupill and stimulate the heat regulating centre situated in floor of third ventricle. The initial stimulation of various centres is followed by depression (Parikh, 1985).

Within half an hour of taking the poison gastric irritation starts. The patients complains of bitter taste, dry mouth and throat,,burning pain in stomach and difficulty in swallowing and talking. This is followed by giddiness, ataxia, inco-ordination of muscles, a peculiar flushed appearance of face, dry hot skin, rise in temperature, diplopia, dilated pupil with loss of accomodation redening of conjunctiva and drowsiness. Some time erythematous rash appears all over body. There is usually full,bounding pulse which later becomes weak and irregular. The patient develops muttering delirium, tries to run away from beds, picks at bed cloths, tries to pull immaginary threads from the tips of his fingers and develops dreadful hallucination of sight and hearing. The condition may pass on to stupor, convulsions, coma and some times death from respiratory failure (Tembe, 1991).

The treatment of Dhatura poisoning is gastric lavage with weak potassium permangnate solution and specific antidote neostigmine given intramuscularly. Excitement and delirium is best controlled with phenobarbitone-sodium administered intramuscularly (Udwadia, 1989).

ALCOHOL POISONING

In general use, the word alcohol means any intoxicating drink of the various alcohols in common use. Only ethyl and methyl alcohols are found in drink, the later only in cheep adultered spirits. The term alcohol is in popular use and is reffered to ethyl alcohol (Ethanol).

Ethyl alcohol depresses the central nervous system irregularly in descending order from cortex to medulla. It first depress the higher centres which control judgement and behaviour (stage of excitement) than the motor centre (stage of incoordination) and finally vital centres in medulla (stage of narcosis). The range between a dose which produce narcosis and one which impairs vital functions is small (Parikh, 1985).

Acute poisoning may result from consumption of an alcoholic beverage in small doses at short intervals or in excessively large dose at a time. The symptoms of acute poisoning is at first a sense of well being, self confidence, flushing of skin and face, a care free behaviour and then gradual loss of self control, argumentativeness, rude behaviour, sentimentality and moresness or melancholic. These are followed by stage of confusion and dulling of perception, muscular incoordination, staggering giat, slurred and incoherent speech, blurred vision and stupor. After a time, recovery may occur accompanied by nausea and vomiting which are regarded as the early signs of recovery. These may be followed by sleeps, severe headache and gastric upset. If recovery does not occurs, the patients passes gradually into unconsciousness, some times hypoglycaemia and coma with slow, stertorous breathing and a full rapid pulse which then becomes slow in volume. The breath smells of alcohol (Modi, 1986).

Occasionally acute alcohol intoxication manifest in atypical patterns, these include in acute excitation which takes form of sudden and unprovoked out burst of anger with even assaultive and destructive behaviour and black outs in the form of episodes of transient amnesia that accompany heavy intoxicants (Gupta, 1988).

As most instances of poisoning in adults are deliberate acts of self poisoning, it is very important that all patients, whether suffering from apparent accidental or intensional poisoning should have a psychiatric assessment. Self poisoning is often important feature of depression in particular. The incidence of repeated acts of self poisoning have been shown clearly to be reduced by early psychiatric consultations (Alexander, 1986).

AIMS OF THE STUDY

1. To study the magnitude of problem of poisoning and its incidence in Bundelkhand region.

2. To study the pattern of poisoning in Bundelkhand region.

3. To study the clinical features of acute poisoning.

4. To study the socio- demographic problem related to poisoning.

M A T E R I A L A N D M E T H O D S

MATERIAL AND METHODS

The present study was conducted in M.L.B. Medical College, Hospital, Jhansi. This study included a combined retrospective and prospective analysis of acute poisoning cases admitted in emergency ward during January, 1990 to December, 1992.

Retrospective study had been carried out from January 1990 to December, 1991. During above two years duration all bed head head ticket of acute poisoning collected from record section, were analysed.

1. Name/address
2. Age/sex/Religion
3. Rural/urban area.
4. Type of poison
5. Cause of poisoning :
   - Suicidal
   - Homicide
   - Accidental
6. Alive/expired/LAMA/Absconded
7. Duration of stay in the hospital
8. Investigations : a. L.F.T.
   b. R.F.T. - Blood urea
      - Serum creatinine.
   c. E.C.G.

Prospective study had been carried out from January, 1992 to December, 1992. During the study every case was subjected to a thorough history and clinical examination as listed below :

HISTORY

The diagnosis of poisoning was based on a reliable history given by patient own or by attendant or remaining tablet or empty container in his/her pocket. In history specific points, recorded like history of psychiatric illness such as depression, socio-economic status, single or joint family, history of drug addiction, failure in examination, termination from job, querells with family members and harrasment by family member or others.

A. Name — Age/Sex

Rural/Urban — Religion/Caste

Marital status — Occupation

Name of poison — Solid/liquid/gaseous form

Type of poisoning — Cause of poisoning

- Suicidal
- Accidental
- Homicidal
- Stupefying

Chief complaints

B. PHYSICAL EXAMINATION

General condition	Icterus
Pulse rate/min	Cyanosis
Blood pressure	Clubbing
Temperature	Hydration
Respiratory rate	Oedema
Pallor	Lymphadenopathy

C. SYSTEMIC EXAMINATION

C.N.S.

a. Conscious/unconscious, if conscious then grade of unconscious according to Edinburgh method :

Grade 0 - Fully conscious

Grade 1 - Drowsy but responsive to vocal commond.

Grade 2 - Unconscious but responsive to minimum painful stimuli.

Grade 3 - Unconscious but just responsive to strong painful stimuli.

Grade 4 - Unconscious with no response to painful stimuli.

b. Pupil - Size

- Light reflex.

c. Motor/sensory

2. C.V.S. (Cardiovascular system)

3. Respiratory system

4. Abdomen.

D. INVESTIGATIONS

1. TLC, DLC, ESR, Hb%

2. Liver function test :

   - Serum bilirubin, SGOT, SGPT, Serum protein.

3. Renal function test

   - Blood urea, serum creatinine.

4. Urine albumin sugar, microscopic examination.

5. Electrocardiography.

E. TREATMENT

a. Medicolegal formalities.

b. Identification of poison, because some poisons have selective antagonist.

c. Removal of poisons by vomiting and gastric lavage.

d. Prevention of further absorption of poison by specific antidote and universal antidote.

e. Forced diuresis.

f. Supportive management :

   i) Airway

   ii) Monitoring of blood pressure,

   iii) Care of back bladder and bowel.

   IV. Nutrition.

g. Haemodialysis.

O B S E R V A T I O N S

OBSERVATIONS

The study consisted of 194 prospective cases of poisoning observed during January, 1992 to December, 1992 and analysis of 251 retrospective cases collected from Medical Record Section admitted during January, 1990 to December, 1991 in M.L.B. Medical College, Hospital, Jhansi was done. Thus the total number of cases analysed became 445.

INCIDENCE

The overall incidence of acute poisoning was 6/1000/year of total hospital admission, but yearly incidence was 5/1000 in 1990, 6/1000 in 1991 and 8/1000 in 1992 as shown in table I.

TABLE I : Incidence of yearly admissions and deaths of acute poisoning cases.

Year	Total No.of hospital admissions	Total deaths	Total admitted cases of poisoning		Total deaths in poisoning cases	
			No.	Incidence/ 1000	No.	Incidence/ 1000
1990	24,031	1,502	116	5	18	12
1991	25,209	1,598	135	6	21	13
1992	25,896	1,690	194	8	43	25
TOTAL	75,136	4,790	445	6	82	17

The incidence of acute poisoning in relation to total emergency admissions and total cases of medicine in emergency admissions is given in table II.

The overall incidence in relation to total emergency admissions was 16/1000/year, but yearly incidence was 12/1000, 15/1000 and 21/1000 in 1990, 1991 and 1992 respectively.

The overall incidence of acute poisoning in relation to total cases of medicine in emergency department was 72/1000/year but yearwise incidence was 61, 67 and 84 per thousand in 1990, 1991 and 1992 respectively. The data indicated that the incidence of acute poisoning has been progressively increasing year by year.

TABLE II : Incidence of poisoning in relation to total admissions in emergency ward and total cases of medicine in the emergency.

Year	Total admission in emergency	No. of medicine cases in emergency	Total cases of poisoning	Incidence/1000 of poisoning in emergency in relation to	
				Total admission	Total cases of medicine
1990	8,992	1,890	116	12	61
1991	9.114	2,005	135	15	67
1992	9,371	2,307	194	21	84
TOTAL	27,477	6,202	445	16	72

RESIDENCE

Out of 445 cases of acute poisoning, 305(68.1%) belonged to Uttar Pradesh and 142 cases belonged to Madhya Pradesh. Out of 116 cases in 1991, 80(69%) cases belonged to UP and 36(31%) cases belonged to M.P., whereas in 1992, out of 135 cases, 91(67.4%) belonged to

U.P. and 44(32.6%) belonged to M.P. and in 1992, out of 194 cases, 132 (68%) belonged to U.P. and 62(32%) belonged to M.P. (Table III).

TABLE III : Incidence of poisoning cases according to their regional back ground.

Year	Uttar Pradesh		Madhya Pradesh		Total
	No.	%	No.	%	No.
1990	80	69.00	36	31.00	116
1991	91	67.40	44	32.60	135
1992	132	68.00	62	32.00	194
TOTAL	303	68.10	142	31.90	445

Table IV shows that 316(71%) cases belonged to rural areas and remaining 129 (29%) cases belonged to urban areas.

Out of 445 cases, 241(54.2%) cases were males and 204(45.8%) cases were females. Overall male : female ratio was 1 : 0.85 and yearwise ratios were 1:0.93, 1:0.87 and 1:0.87 in 1990, 1991 and 1992 respectively (Table IV).

TABLE IV : Distribution of cases according to sex and rural & urban areas.

Year	Rural		Urban		Total		M : F ratio
	Male	Female	Male	Female	Male	Female	
1990	45	43	15	13	60	56	1 : 0.93
1991	58	40	19	18	77	58	1 : 0.87
1992	70	60	34	30	104	90	1 : 0.87
TOTAL	173	143	68	61	241	204	1 : 0.85
	316		129				

AGE AND SEX

TABLE V : Distribution of cases according to their age and sex.

Age group (years)	Male		Female		Total	
	No.	%	No.	%	No.	%
≤ 1	4	1.70	4	2.00	8	1.80
2 - 4	24	10.00	14	6.90	38	8.50
5 - 9	24	10.00	18	8.80	42	9.50
10 - 14	9	3.70	4	2.00	13	2.91
15 - 24	94	39.00	91	44.60	185	41.60
25 - 34	65	27.00	50	24.50	115	25.80
35 - 44	16	6.60	10	4.90	26	5.80
45 - 54	2	0.80	6	2.90	8	1.80
55 - 64	2	0.80	6	2.90	8	1.80
7 65	1	0.40	1	0.50	2	0.50
TOTAL	241	100.00	204	100.00	445	100.00

Table V shows that 8(1.8%) cases were below the 1 year of age. 38(8.5%) cases belonged to 2-4 years age group, 42(9.5%) cases belonged to 5-9 years of age group, 13(2.9%) cases belonged to 10-14 year of age group, 185 (41.6%) cases belonged to 15-24 years of age group, 115 (25.8%) cases belonged to 25-34 years age group, 26(5.8%) belonged to 35-44 years and 8(1.80%) cases belonged to 45-54 and 55.64 years of age group each and there were only two (0.5%) cases more than 65 years of age.

Table V shows that maximum 185(41.6%) cases

belonged to 15-24 years of age followed 115(25.8%) cases belonging to 25-34 years of age. The minimum two cases (0.5%) belonged to more than 65 years of age group. Majority (300, 67.4%) of the cases belonged to 15-34 years of age group.

RELIGION

Table VI shows that out of 445 cases, 379(85.2%) cases were Hindus followed by 49(11.0%) cases of Muslim community. Whereas 10(2.2%) cases were Sikh and 5(1.1%) cases were Christians. Religion was unknown in 2(0.2%) cases.

TABLE VI : Distribution of cases according to their religion.

Religion	No. of cases	Percentage
Hindu	379	85.20
Muslim	49	11.00
Sikh	10	2.20
Christian	5	1.10
Unknown	2	0.50
TOTAL	445	100.00

MARITAL STATUS AND TYPE OF POISONING

Table VII shows that out of 445 cases of poisoning, 236(53%) cases were married whereas 209(47%) cases were unmarried. Maximum 336(75.5%) cases were suicidal followed

by 88(19.8%) cases were accidental and the minimum 5(1.1%) cases were homicidal and 16(3.6%) cases were of stupefying poisoning.

TABLE VII : Distribution of cases according to their marital status and type of poisoning.

Type of poisoning	Marital status				Total	
	Married		Unmarried			
	No.	%	No.	%	No.	%
Suicidal	183	77.50	153	73.20	336	75.50
Accidental	38	16.10	50	23.90	88	19.80
Stupefying	15	6.40	1	0.50	16	3.60
Homicidal	-	-	5	2.40	5	1.10
TOTAL	236	53.00	209	47.00	445	100.00

Table VII also shows that out of 236 married cases, 183(77.5%) cases were suicidal, 38(16.1%) cases were accidental and 15(6.1%) cases were stupefying and in unmarried cases, 153 (73.2%) cases were suicidal, 50 (23.90%) were accidental and 5(2.4%) cases were homicidal. Only 1(0.5%) case was stypefying poisoning.

SOCIO-ECONOMIC STATUS

Table VIII shows that out of 194 prospective cases, 143(73.7%) cases belonged to low socio-economic group followed by 36(18.6%) cases belonging to middle socio-economic group and 15(7.7%) cases belonged to upper class.

TABLE VIII : Distribution of 194 prospective cases according to their socio-economic status.

Socio-economic Status	Poisoning cases	
	No.	Percentage
Upper class	15	7.70
Middleclass	36	18.60
Lower class	143	73.70
TOTAL	194	100.00

FAMILY BACKGROUND AND TYPE OF POISONING

TABLE IX : Distribution of cases according to their family and type of poisoning.

Type of poisoning	Type of Family				TOTAL	
	Nuclear		Combined		No.	%
	No.	%	No.	%		
Suicidal	70	82.30	91	83.50	161	83.00
Accidental	13	15.30	14	12.80	27	13.90
Stupefying	1	1.20	3	2.80	4.	2.10
Homicidal	1	1.20	1	0.90	2	1.00
TOTAL	85	43.80	109	56.20	194	100.0

Table IX shows that out of total 194 prospective cases of poisoning, 109(56.2%) cases were belonging to joint family. Out of which, in 91(83.5%) cases the poisoning was suicidal and in 14(12.8%) cases it was accidental. There were 85(43.80%) cases were of nuclear families. Out of which, in 70 (82.3%) cases the poisoning was suicidal and in 13(15.3%) cases it was accidental.

TABLE X : Year wise distribution of poisoning cases according to their type of poisoning.

Year	Total cases	TYPE OF POISONING IN EACH YEAR							
		Suicidal		Accidental		Stupefying		Homicidal	
		No.	%	No.	%	No.	%	No.	%
1990	116	86	74.1	26	22.4	3	2.6	1	0.9
1991	135	97	72.0	29	21.5	7	5.0	2	1.5
1992	194	153	78.8	33	17.0	6	3.2	2	1.0
TOTAL	445	336	75.5	88	19.8	16	3.6	5	1.1

Table X shows that out of 445 cases, 336(75.5%) cases were suicidal and 88(19.8%) cases were accidental. 16(3.6%) cases were stupefying poisoning and 5 cases were homicidal. Out of 116 cases of 1990, 86(74.1%) cases were suicidal, 26(22.4%) cases were accidental, 3(2.6%) cases were stupefying and 1 case was of homicidal poisoning. In 1991, out of 135 total cases, 97(72%) cases were suicidal, 29(21.5%) cases were accidental, 7(5%) cases were of stupefying poisoning and 2(1.5%) cases were of homicidal poisoning and in 1992, out of 194 cases of poisoning, 153 153(78.8%) cases were of suicidal poisoning, 33(17%) cases were accidental, 6(3.2%) were of stupefying poisoning and 2(1%) cases were of homicidal poisoning.

MONTHLY DISTRIBUTION OF CASES.

Table XI shows that out of 445 cases, 57(12.8%) cases were admitted in the month of June followed 51(11.5%) cases who were admitted in month of April. The minimum 26 (5.8%) cases were admitted in the month of March. The

majority (221, 49.7%) cases were admitted from April to July.

TABLE XI : Distribution of poisoning cases according to month of their admission in the hospital.

Month	1990		1991		1992		Total	
	No.	%	No.	%	No.	%	No.	%
January	6	5.10	16	11.90	10	5.20	32	7.20
February	9	7.80	12	8.90	15	7.70	36	8.00
March	7	6.00	7	5.20	12	6.20	26	5.80
April	14	12.10	14	10.40	23	11.90	51	11.50
May	11	9.50	20	14.80	16	8.20	47	10.60
June	15	12.90	16	11.90	26	13.40	57	12.80
July	14	12.10	10	7.40	22	11.30	46	10.30
August	8	6.90	7	5.20	14	7.20	29	6.50
September	9	7.80	5	3.50	17	8.80	31	7.10
October	8	6.90	7	5.20	15	7.70	30	6.70
November	8	6.90	7	5.20	17	8.80	32	7.20
December	7	6.00	14	10.40	7	3.60	28	6.30
TOTAL	116	100.00	135	100.0	194	100.0	445	100.0

PATTERN OF POISONING

Overall pattern of poisoning was aluminium phosphide -91(20.5%) cases, dhatura in 67(15%) cases, rat killer in 64(14.4%) cases, organophosphorus compound in 61(13.7%) cases, alcohols in 41(9.3%) cases, sedative such as morphine, phenobarbitone, diazepam etc. in 29(6.5%) and unknown poisoning was in 83(7.6%) cases and in

miscellaneous group such as petroleum (kerosine, diesel) in 14(3.4%) cases, alkalies and acids in 8(1.8%), copper sulphate in 8(1.8%) and drugs such as isonex, largactil, mixed etc. in 27(6%) cases. The commonest poisoning was aluminium phosphide in 20.5% cases and yearly distribution it was in 12% cases in 1990, 17.8% cases in 1991 and in 1992 it was in 27.3% cases. It is evident from table XII that there was an increasing trend year by year (Table XII).

TABLE XII : Pattern of poisoning in three years.

Name of poison	1990		1991		1992		Total	
	No.	%	No.	%	No.	%	No.	%
Aluminium phosphide	14	12.0	24	17.8	53	27.3	91	20.5
Dhatura	21	18.1	23	17.0	23	11.8	67	15.0
Rodenticide (Rat killer)	20	17.2	22	16.3	22	11.3	64	14.4
Organophosphorus compound	16	13.8	15	11.1	30	15.5	61	13.7
Alcohols	11	9.5	9	6.7	21	10.8	41	9.3
Sedatives: morphine phenobarbitone, diazepalm etc.	9	7.8	11	8.2	9	4.6	29	6.5
Unknown	11	9.5	10	7.4	14	7.2	33	7.6
Miscellaneous:								
- Petroleum (kerosene, diesel)	4	3.4	3	2.2	7	3.6	14	3.4
- Alkalies & acids	1	0.9	4	2.9	3	1.6	8	1.8
- Copper sulphate	2	1.7	4	2.9	2	1.0	8	1.8
- Drugs	7	6.1	10	7.5	10	5.3	27	6.0
TOTAL	116	100.0	135	100.0	194	100.0	445	100.0

CLINICAL FEATURES

TABLE XIII : Clinical features of cases in major poisoning groups.

Clinical features	NAME OF THE POISON TAKEN					
	Aluminium phosphide (N=91)		Organoph-osphorus (N=61)		Dhatura (N=67)	
	No.	%	No.	%	No.	%
Gastrointestinal upset (Nausea, vomiting, pain)	88	96.7	43	70.5	37	55.2
Shock (Systolic BP ⟨90)	65	71.4	7	11.5	2	3.0
Tachycardia, sweating	67	73.6	12	19.7	51	76.1
Restlessness, altered sensorium, drowsyness	44	48.4	31	50.8	57	85.1
Tachyapnoea, dyspnoea, frothing from mouth, Johnchi, crepts	28	30.8	41	67.2	15	22.4
Fever	2	2.2	-	-	17	25.4
Oliguria	6	6.6	2	3.2	-	-
Bradycardia	16	17.6	30	49.2	-	-
S_3/S_4 gallop rhythm	21	23.1	-	-	-	-
Seizure	2	2.2	3	4.9	-	-
Tenderhepatomegaly	5	5.5	-	-	-	-
Pupils - Constricted	-	-	35	57.4	-	-
- Semidilated	50	54.9	2	3.2	-	-
- Dilated	2	2.2	-	-	42	62.7

The clinical features of major group of poisoning has been shown in table XIII.

In most of the aluminium phosphide cases the signs and symptoms were gastrointestinal upset (88, 96.7%) (nausea, vomiting and pain etc.), in 69(73.6%) cases there was tachycardia, shock (systolic blood pressure ⟨90 mm Hg) was in 65(71.4%) cases, semidilated pupils were present in 50

(54.9%) cases, restlessness was in 44(48.4%) cases, dyspnoea and tachypnoea were in 28(30.8%) cases, gallop rhythm and bradycardia were in 21(23.1%) and 16(17.6%) cases respectively. Oliguria, tenderhepatomegaly, seizure and fever were present in lesser number of cases.

In most of the cases of organophosphorus poisoning gastrointestinal upset was present in 43(70.5%) cases, dyspnoea, tachyapnoea and frothing from mouth were found in 41(67.2%) cases, constricted pupils were in 35(57.4%) cases. Restlessness and altered sensorium were found in 31(50.8%) cases, bradycardia was in 30(49.2%) cases and tachycardia, shock, oliguria, seizure, semidilated pupils were present in lesser number of cases.

In Dhatura poisoning, restlessness altered sensorium, drowsiness and delirium were present in 57 (85.1%) cases, Tachycardia, dilated pupil, gastrointestinal upset and pyrexia were found in 51(76.11%), 42(62.7%), 37(55.2%) and 17(25.4%) cases respectively.

HOSPITAL STAY

Our of 445 cases, 297 (66.7%) cases were stayed in the hospital upto 3 days, whereas 96(21.6%) cases were stayed for 4-6 days. 27(6.1%) cases stayed for 7-8 days and remaining 25(5.6%) cases stayed in the hospital for 9 days or more (Table XIV).

TABLE XIV : Distribution of cases according to their hospital stay and poisoning.

Name of poison	No.of cases	HOSPITAL STAY (DAYS) 0-3 No.	0-3 %	4-6 No.	4-6 %	7-8 No.	7-8 %	9 No.	9 %
Aluminium phosphide	91	70	76.9	18	19.8	3	3.3	-	-
Rodenticide	64	45	70.3	15	23.4	4	6.3	-	-
Dhatura	67	41	61.2	22	32.8	2	3.0	2	3.0
Organophos-phorus compounds	61	33	54.1	11	18.4	10	16.4	7	11.1
Alcohols	41	31	75.6	10	24.4	-	-	-	-
Sedatives	29	10	34.5	12	41.4	4	13.8	3	10.3
Miscella-neous	92	67	72.8	8	8.7	4	4.3	13	14.2
TOTAL	445	297	66.7	96	21.6	27	6.1	25	5.6

INVESTIGATIONS

E.C.G. CHANGES

Out of 111 prospective cases, 50(45.1%) cases showed ECG changes. Out of these 50 cases, in 31(62%) cases ECG changes were occurred due to aluminium phosphide, 7(14%) cases each were of rat killer and organophosphorus 4(8%) cases were of ethylene dibromide (EDB) and remaining 1(2%) case was of dhatura poisoning.

Out of 39 cases of aluminium phosphide poisoning, 31(79.5%) cases showed ECG changes, Out of 24 rat killer cases, 7(29.2%) , out of 27 organophosphorus poisoning, 7(25%) cases, out of 11 cases of Dhatura poisoning 1(11.1%)

cases and all 4 cases of ethylene dibromide cases showed positive ECG changes. No ECG changes were observed in alcohol, sedatives and miscellaneous poisonings.

In aluminium phosphide poisoning group, the commonest ECG finding was ST-T changes (ST segment depression or elevation with T wave inversion). This was observed in 26(66.7%) cases. The other ECG changes were atrial fibrillation in 10(25.6%) cases, sinus tachycardia in 9(23.1%) cases, ventricular ectopics and SVT in 7 (17.9%) cases each, bradycardia and LBBB in 5(12.8%) each, RBBB in 4(10.3%) and wandering pacemakers was present in 1 (2.6%) case.

The commonest ECG changes were ST-T changes in 6(25%) cases in rat killer poisoning and other uncommon were atrial fibrillation, bradycardia and RBBB in 2(8.3%) cases each and sinus tachycardia and LBBB was present in 1(4.2%) case.

In organophosphorus poisoning, the commonest ECG changes were bradycardia in 5(18.5%) cases. Other changes were SVT in 3(11.1%) cases and sinus tachycardia in 2(7.4%) cases (Table XVI).

TABLE XV : ECG changes in different poisoning groups.

Name of poison	Total cases	Positive ECG changes No. (%)	Percentage
Aluminium phosphide	39	31(62)*	79.5
Rat killer(Rodenticide)	24	7(14)	29.2
Organophosphorus compounds	27	7(14)	25.9
Dhatura	9	1(2)	11.1
Alcohols	3	-	
Sedatives	3	-	
Miscellaneous : EDB	4	4(8)	100.00
: Copper Sulphate	2	-	
TOTAL	111	50(45.1%)	100.00

* Percentage given in parantheses based on total number of cases in which ECG changes present.

TABLE XVI : Type of ECG changes observed in major poisoning groups.

ECG Changes	NAME OF POISONS					
	Aluminium phosphide		Rodenti-cide		Organopho-sphorus	
	No.	%	No.	%	No.	%
ST-T changes(elevation or depression)	26*	66.7	6	25.0	-	-
Atrial fibrillation	10	25.6	2	8.3	-	-
Sinus tachycardia	9	23.1	1	4.2	2	7.4
Ventricular ectopics	7	17.9	-	-	-	-
Supra ventricular tachycardia	7	17.9	1	4.2	3	11.1
Bradycardia	5	12.8	2	8.3	5	18.5
RBBB	4	10.3	2	8.3	-	-
LBBB	5	12.8	1	4.2	-	-
Wandering pacemaker	1	2.6	-	-	-	-
No change	8	20.5	17	70.8	20	74.1

* There may be present more than one abnormalities

BLOOD INVESTIGATIONS

Table XVII shows changes in blood in some cases of common poisoning..

Aluminium phosphide Poisoning

Out of 91 cases of aluminium phosphide poisoning, 31 cases were investigated for blood urea, serum creatinine. Out of these 31 cases, 3(9.7%) cases had raised values of blood urea and serum creatinine, and 23 patients were investigated for serum bilirubin, SGOT and SGPT, in which serum bilirubin was raised in 3(13%) cases and SGOT & SGPT were raised in 7(30.4%) each.

Rat killer poisoning

Out of 64 cases of rat killer poisoning, 14 were investigated for blood urea and serum creatinine, out of these 14 cases, blood urea and serum creatinine were raised in 1 (7.1%) case each. and serum bilirubin, SGOT & SGPT were investigated in 9 cases each. Out of these 9 cases serum bilirubin was raised in 1(11.1%) cases, and SGOT and SGPT were raised in 2 (22.2%) cases each.

Organophosphorus poisoning

Out of 61 cases of this group of poisoning, enzyme SGPT, SGOT and serum bilirubin were investigated in 11 cases each and these were found to be raised in 3(27.3%), 3(27.3%) and 1(9.1%) cases respectively.

Miscellaneous :Ethelene dibromide (EDB)

It is evident from the table XVII that the blood

urea and serum creatinine were raised in all four cases (100%) studied for these tests. Similarly enzymes SGPT, SGOT and serum bilirubin were also increased in 3(75%) cases each out of 4 cases in which these tests were performed.

TABLE XVII : Results of blood investigations in poisoning cases.

Investigation performed	No.of cases studied	Results of investigations			
		Normal values		Raised values	
		No.	%	No.	%
ALUMINIUM PHOSPHIDE					
Blood urea	31	28	90.3	3	9.7
Serum creatinine	31	28	90.3	3	9.7
Serum bilirubin	23	20	87.0	3	13.0
S.G.O.T.	23	16	69.6	7	30.4
S.G.P.T	23	16	69.6	7	30.4
RAT KILLER(RODENTICIDE)					
Blood urea	14	13	92.9	1	7.1
Serum creatinine	14	13	92.9	1	7.1
Serum bilirubin	9	8	88.9	1	11.1
S.G.O.T.	9	7	77.8	2	22.2
S.G.P.T.	9	7	77.8	2	22.2
ORGANOPHOSPHORUS					
Blood urea	18	18	100.0	-	-
Serum creatinine	18	18	100.0	-	-
Serum bilirubin	11	10	90.9	1	9.1
S.G.O.T.	11	8	72.7	3	27.3
S.G.P.T.	11	8	72.7	3	27.3
MISCELLANEOUS (EDB)					
Blood urea	4	-	-	4	100.0
Serum creatinine	4	-	-	4	100.0
Serum bilirubin	4	1	25.0	3	75.0
S.G.O.T.	4	1	25.0	3	75.0
S.G.P.T.	4	1	25.0	3	75.0

MORTALITY

The overall incidence of deaths of acute poisoning was 17/1000/year of total hospital deaths, but yearly incidence was 12/1000 in 1990, 13/1000 in 1991 and 25/1000 in 1992 (Table I).

Out of 445 cases, 316(71%) cases survived, 82(18.4%) expired and remaining 47(10.6%) cases left the hospital against medical advice or absconded.

Out of total 82 deaths, 56(68.3%) deaths were due to aluminium phosphide poisoning, 12(14.6%) death were due to organophosphorus poisoning, 4(4.9%) deaths were due to rat killer, 3(3.7%) were due to alcohols and miscellaneous poisoning each, 2(2.4%) deaths were due to dhatura poisoning and 1(1.2%) case each was due to sedative and unknown poisoning.

Out of total 91 cases of aluminium phosphide poisoning 56(61.5%) cases expired, 27(29.7%) cases survived and remaining 8(8.8%) cases left the hospital against medical advice or absconded.

Out of 61 cases of organophosphorus poisoning, 44(72.1%) cases survived, 12(19.7%) expired and remaining 9(13.4%) cases absconded or left against medical advice.

In other poisoning groups i.e. rat killer, dhatura, alcohol, sedatives and unknown and miscellaneous approximately 76-90% cases survived, 3-7% cases expired and 5-15% cases left the hospital against medical advice or absconded (Table XVIII).

TABLE XVIII : Poisoning cases and outcome of treatment.

Name of poison	Total cases studied	OUTCOME OF TREATMENT					
		Survived		Expired		Absconded/ LAMA	
		No.	%	No. (%)	%	No.	%
Aluminium phosphide	91	27	29.7	56(68.3)	61.5	8	8.8
Dhatura	67	56	83.6	2(2.4)	3.0	9	13.4
Rat killer	64	57	89.1	4(4.9)	6.2	3	4.7
Organophosphorus	61	44	72.1	12(14.6)	19.7	5	8.2
Alcohol	41	31	75.6	3(3.7)	7.3	7	17.1
Sedatives	29	26	89.7	1(1.2)	3.4	2	6.9
Unknown	33	27	81.8	1(1.2)	3.3	5	14.9
Miscellaneous	59	48	82.5	3(3.7)	3.5	8	14.0
TOTAL	445	316	71.0	82(100)	18.4	47	10.6

Percentage give in parantheses are based on total deaths.

Table XIX shows that out of 91 cases of aluminium phosphide poisoning, 29(31.9%) cases took $\leq$ 1 tablet, 20(22%) cases took 2 tablets and 21(23.1%) cases took 3 tablets and $\geq$6 tablets were taken by 9(9.91%), 7(7.7%) and 5(5.4%) cases respectively. The higher mortality rate (66.7 to 85.7%) was in those patients who were taking 3 or more tablets. In patients taking $\leq$ 2, mortality rate was 45 - 51.7%(Table XIX).

A total number of 53 cases of aluminium phosphide poisoning, 42(79.2%) cases were taking non exposed (fresh) tablets, while remaining 11(20.8%) patients had consumed

exposed (used) tablets (Table XX).

TABLE XIX : Outcome of treatment in cases of aluminium phosphide poisoning according to quantity of tablet intake.

No. of tablets taken	Total cases Studied		OUTCOME OF THE TREATMENT					
			Survived		Expired		Absconded/ LAMA	
	No.	%	No.	%	No.	%	No.	%
≤1	29	31.9	10	34.5	15	51.7	4	13.8
2	20	22.0	10	50.0	9	45.0	1	5.0
3	21	23.1	5	23.8	16	76.2	-	-
4	9	9.9	1	11.1	6	66.7	2	22.2
5	7	7.7	1	14.3	6	85.7	-	-
≥6	5	5.4	-	-	4	80.0	1	20.0
TOTAL	91	100.0	27	29.7	56	61.5	8	8.8

TABLE XX : Outcome of treatment in cases of aluminium phosphide poisoning according to tablets (exposed/fresh) used.

Tablet taken	Total cases studied		OUTCOME OF TREATMENT					
			Survived		Expired		Absconded/ LAMA	
	No.	%	No.	%	No.	%	No.	%
Exposed	11	20.8	8	73.7	2	18.2	1	9.1
Nonexposed (afresh)	42	79.2	12	28.6	26	61.9	4	9.5
TOTAL	53	100.0	20	37.7	28	52.9	5	9.4

Out of 42 cases of aluminium phosphide poisoning taking nonexposed (afresh) tablets, 12(28.6%) cases survived, 26(61.9%) cases expired and remaining 4 cases

(9.5%) left against medical advice or absconded.

Out of 11 cases using exposed (used) tablets, 8(73.7%) cases survived, 2(18.2%) expired and 1(9.1%) case absconded from the hospital or left the hospital against medical advice.

The above data indicate that mortality rate is more in those patients who took nonexposed (afresh) tablets as compared to those patients who took exposed (used) tablets.

PSYCHIATRIC ASSESSMENT

A total of 87 prospective cases studied for psychiatric assessment, 8(9.2%) cases had psychiatric illness. Out of these 8 cases of psychiatric disease, 6 cases were suffering from depression and 2 were suffering from schiezophrenia.

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D I S C U S S I O N

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DISCUSSION

The present study consisted of 445 cases of acute poisoning, admitted in the hospital from January 1990 to December, 1992. The overall incidence of acute poisoning was 6/1000/year of total hospital admissions. The overall incidence in relation to total emergency admissions was 16/1000/year whereas it was reported by Kumar et al (1989) as 21.5/1000. In emergency roughly every 13th case on medical side was of acute poisoning. Incidence of poisoning has been increasing year by year possibly because of increasing incidences of marital dishormony, broken engagements, harrassment by family members, various family problems, unemployment, loss of job, failure in examination, depression, prolonged illness and personality disorders.

TYPES OF POISONING

The intension was suicidal in 75.5% cases, accidental in 19.8% cases, stupefying in 3.6% and remaining 1.1% cases were homicidal. Almost similar type of results have been observed viz. suicidal (50-97%), accidental (3-26%), stupefying (0-21%) and homicidal (0-20%) by Siwatch et al (1988), Kumar et al (1989), Chug et al (1991), Mahapatra et al (1991) and Khosla et al (1992).

Accidental poisoning was most common (88%) in childhood, because they starts crawling and walking at the

age of 1 year and are very active and try to explore unfamiliar objects by putting into their mouth(oral phase of psychosexual development). Thus they exposed themselves to accidental poisoning. Other etiological factors may be large family, in which mother is often engaged with household works, carelessness in storage of the poisonous substances, due to little storage facilities in small houses, poisonous substances are stored in easily accessible places, hence children living in these small and over crowded houses are more exposed to accidental poisoning.

Stupefying poisoning occurred mostly for robbery purposes and rarely for kidnepping and rape.

RESIDENCE AND RELIGION

In present study, 68.1% cases of poisoning belonged to Uttar Pradesh and remaining 31.9% belonged to Madhya Pradesh. It is because this medical college is situated in the border of UP and MP both. A total 316 (71%) cases belonged to rural areas and remaining 129(29%) belonged to urban areas. According to 1981 census, the percentage of rural and urban population was 73 and 27 respectively. Considering this, there is hardly any difference in the incidence of poisoning in urban and rural areas. Multani et al (1991) reported slightly higher incidence of poisoning in urban (34.5%) and 65.5%) in rural areas. Our findings are in conformity with the respect of Chug et al (1991), who reported the incidence

in rural area 85% and urban 15%.

Eighty five percent of total poisoning cases were Hindus. According to 1981 census, population of Hindus was 78.2%. Thus, incidence of poisoning is slightly higher in proportion to population of Hindus, but this difference is not likely to be statistically significant in overall.

There were 271 (54.2%) males and 204(45.8%) cases females in the present study. The Male : Female ratio was 1 : 0.85. According to 1991 census male:female ratio was 1 : 0.93. Thus poisonings are almost same in males and females. Almost similar type of results i.e. 46.3 to 78.6% males and 21.4 to 53.7% females were observed by Kumar et al (1989), Samaria et al (1990), Multani et al (1991) and Mahapatra et al (1991).

Out of 445 cases, 185(41.6%) cases belonged to age group of 15-24 years, 115(25.8%) cases belonged to 25-34 years of age, and 46(10.3%) cases belonged to 0-4 years of age group. In 1991 census 18.2% population belonged to 15-24 years of age group, 14% belonged to 25-34 years and 12.6% belonged to 0-4 years of age. Thus, poisoning was two times more common in age group of 15-24 and 25-34 years of age group and in children the poisoning was more common in age group of 0-4 years of age as compared to proportion of population of 1991 census. It was because of children in this age group try to explore

unfamiliar objects by putting into mouth. Previous studies showed that 73.3% cases belonged to 2nd decade (Multani et al, 1991) and 66.1% in 2nd and 3rd decade (Mahapatra et al, 1991).

SOCIO-ECONOMIC AND MARITAL STATUS

In the present study, 56.2% cases of acute poisoning belonged to joint (combined) family and 53% cases were married. 73.7% cases were from low socio-economic group. Kumar et al (1989) reported 56.36% cases from low socio-economic group. This higher incidence in present study was due to much more poverty striken population in Bundeklhand region.

MONTHLY DISTRIBUTION

Majority of the cases of acute poisoning admitted in the month of June (57, 12.8%). Around 201 (45.2%) cases admitted between the months of April to July. This increase in incidence of poisoning during these months was because, most of the results (high school, intermediate, graduate and postgraduate) are declared during these months, and persons who do not succeed in examinations, some of them take poisons. During these months corn is also stored with aluminium phosphide tablets, EDB ampoule are put into stored corn to repel or kill the rodents and other insects.

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PATTERN OF POISONING

In the present study, aluminium phosphide was commonest (91, 20.5%) poisoning followed by Dhatura(61, 15%), rodenticide (14.4%), organophosphorus compounds and other remaining were miscellaneous group.

In 1990, aluminium phosphide poisoning was 12% but progressively increased and reached to 27.3% in 1992. It is commonest suicidal agent in northern India as reported by Bajaj et al (1988). Chug et al (1991) described that aluminium phosphide in Haryana was unknown before 1980, but it increased progressively and surpassed other poisonings in Haryana. Incidence of aluminium phosphide poisoning is progressively increasing in whole of northern India. Recently patients of aluminium phosphide reported from Chandigarh by Singhal et al (1985), Jain et al (1988) from M.P., Bajaj et al (1988) from Delhi and Ram et al (1988) & Mishra et al (1991) from Uttar Pradesh. Aluminium phosphide poisoning was commonest because it is very cheap and easily available in market and more effective.

Other major groups of poisoning were dhatura (15%), rat killer (rodenticide) (14.4%), organophosphorus compounds (61, 13.7%) and other poisonings were 35(7.6%). Dhatura poisoning was common in the use for stupefying to robbery and kidnaping. This poisoning is used during journey.

Most (66.7%) of the poisoning cases were discharged within 3 days from admission. Almost similar

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Most (66.7%) of the poisoning cases were discharged within 3 days from admission. Almost similar

results were reported by Multani et al (1991). They reported that 70% of patients were discharged within 48 hours. Cases of sedative and dhatura poisoning stayed in hospital slightly longer duration because in this poisoning nervous system is affected.

A total of 87 cases were analysed for psychiatric assessment in which 6(6.9%) cases were depressive and 2(2.3%) cases were scheizophrenic (behavioural disorders). Self poisoning is often an important feature of various psychiatric disorder and of depression in particular. (Alexander, 1986).

ALUMINIUM PHOSPHIDE POISONING

The incidence of aluminium phosphide poisoning increases year by year. It was the commonest (20.5%) poisoning in present study. Most of the patients presented in hospital with clinical feature of gastrointestinal upset (nausea, vomiting, burning pain etc). (96.7%), tachycardia - 73.6%, severe hypotension and very feeble and impalpable peripheral pulses - 71.4%, semidilated pupils - 54.9% and restlessness in 48.4% of cases. Almost similar type of clinical feature observed by Siwatch et al (1988), Chug et al (1991) and Khosla et al (1992).

The positive electrocardiographic changes occurred in 79.5% cases of aluminium phosphide poisoning. Previous authors showed that electrocardiographic changes occurred in 36 to 90.8% of acute aluminium phosphide cases.

Puraman et al (1988), Chopra et al (1989) and Khanijoo et al (1991). Positive electrocardiographic changes were ST-T changes (either elevation or depression) - 66.7%, other changes were atrial fibrillation, sinus tachycardia, ventricular ectopics, supraventricular tachycardia, bradycardia, .Right or left bundle branch block. Same type of electrocardiographic changes observed by Raman et al (1985), Sepaha et al (1985), Jain et al (1985), Agarwal et al (1989) and Khosla et al (1992).

On blood investigations enzymes aminotransferases (SGOT, SGPT) increased in 30.4% cases, serum bilirubin raised in 13% and blood urea and serum creatinine were raised in 9.7% cases. These increased value sdepended upon damage of heart, liver and kidney. According to Puraman et al (1989), 84% of aluminium phosphide cases have increased SGOT, SGPT and 44% cases had increased serum creatinine.

76.2% to 100% cases expired, who had taken three tablets or more. Mortality was lower (45-51.7%) in patients who had taken equal or less than 2 tablets. Mortality rate was higher (62%) in those patients who took fresh tablets than the patients who took exposed tablets (20.8%) because in exposed tablets phosphine gas liberated an exposure to atmospheric moisture. Almost similar type of results observed by Singh et al (1985), Dasohara et al (1985), Siwatch et al (1988) and Katira et al (1990).

Patients who had resistant hypotension (not responded to dopamine) associated with oliguria, development of coma, severe hypoxia and chest infection had poor prognosis. Almost same type of bad prognostic indices were showed by Chug et al (1990, 1992).

After giving proper supportive treatment and injection magnesium sulphate, 27(29.7%) cases survived, 56(61.5%) expired and remaining 8(8.8%) cases absconded. So mortality rate in aluminium phosphide poisoning was 61.5%. Previous authors reported mortality ranging from 37 to 100% (Singh et al, 1985; Chopra et al, 1986; Khosla et al, 1988; Ram et al, 1988; Agarwal et al, 1989; Singh et al, 1989 and Katira et al, 1990).

Ninety percent deaths of aluminium phosphide occurred in 1st 24 hours. Similar type of results (93%) were reported by Khosla et al (1992) in aluminium phosphide poisoning in 1st 24 hours.

The aluminium phosphide poisoning was common in agricultural belt. It is emerged as a dangerous weapon to human lives due to its easy availability in homes, easily availability from market and very cheap. Very high mortality in this poisoning is because it is very toxic and damaged to heart, liver, lungs and have no specific antidote.

RODENTICIDE (RAT KILLER) POISONING

It was the third commonest type of poisoning in present study. It comprised 64(14.4%) cases. Most of rodenticide poisoning patients presented with clinical feature of gastrointestinal upsets, tachycardia, semi-dilated pupils, dyspnoea etc.

29.2% of rodenticide poisoning had positive electrocardiographic changes. They were St-T change(25%), atrial fibrillation, bradycardia, right bundle branch block. 22.2% of poisoning cases had raised values of SGOT, SGPT, 11.1% cases had increased value of serum bilirubin and blood urea and serum creatinine were raised in 7.1% cases.

Mortality rate was 6.2%. Almost same type of results were observed by Subramaniam (1990), in which mortality rate was 8.8%. Rodenticide poisons available in the market in the form of powder pouch, it contains ether barium carbonate, or zinc phosphide or warfarin etc. Mortality rate was lesser because of in market fake rodenticide are available. These are less toxic except zinc phosphide.

ORGANOPHOSPHORUS POISONING

A total of 61 (13.7%) cases of organophosphorus poisoning were analysed for various clinical parameters. The substance which were taken by the patients, were malathion, parathion, diazenon (Tick-20) and baygon (propoxor). Previously it was the commonest poisoning

all over India, but its place has been taken by aluminium phosphide poisoning. Previous studies showed that organophosphorus poisoning was 10.3 to 44.5% of total admitted cases of poisoning (Kumar et al, 1989; Mahapatra et al, 1990 and Multani et al, 1991). In present study it was still 4th commonest group of poisoning.

Most of cases of organophosphorus poisoning presented with clinical feature of gastrointestinal upsets(70.5%), respiratory distress (67.2%) and constricted pupils(57.4%). Other clinical features were restlessness, altered sensorium bradycardia, shock, oliguria, convulsions etc. Almost similar type of clinical features were observed by Vishwanathan et al (1962), Gupta et al(1968), Singh et al (1969), Natarajan (1977), Gupta et al. (1990) and Bitchile et al (1990).

The positive electrocardiographic changes were observed in 25.9% cases. Commonest electrocardiographic changes were bradycardia (18.5%), Supraventricular tachycardia (11.1%) cases. Almost similar type of results were observed by Gupta et al (1990) and Singh et al (1991). They reported 10-30% cases of organophosphorus poisoning have positive ECG changes. On blood investigation SGOT, SGPT raised in 29.3% cases and serum bilirubin raised in 9.1% cases.

19.7% cases of organophosphorus poisoning expired. Almost similar mortality rate (18.4 to 43.0%)

was observed by Bhakat ram et al (1990), Chammundesh Swami (1990), Mullani et al (1990) and Ghosh et al (1991).

Organophosphorus poisoning was common because of it extensively used as pesticide in agriculture, some of these substances are in common domestic used for destruction of vermin or rodents. The public has no difficulty in obtaining them, when the impulse to commit suicide arises, they are ready at hand. Lack of awareness regarding to methods of spraying caused organophosphorus compound poisoning in large number of cases, which can be prevented by proper education with the help of mass media.

DHATURA POISONING

It was second commonest group of poisoning in our study, it comprises 67 (15%) cases of dhatura poisoning. The clinical presentation were restlessness, drowsy and altered sensorium 85.1% cases, tachycardia in 76.1%, dilated pupils (62.7%) and gastrointestinal upsets in 55.2% cases. No positive electrocardiographic changes were observed except tachycardia. The mortality rate was 3%.

It was common poisoning because in rural area it is easily available, it is employed mainly as stupefying poison, mostly for purpose of robbery. Some people takes it as prasad of God Shivji and some people use it as an aphrodisiac. Accidental cases also occurred in children and adults, who eats raw fruits or seeds mostly from edible

fruits or capsicum seeds respectively. Accidental cases also occurred from the use of dhatura seeds by quacks for treatment of various ailments.

IN MISCELLANEOUS GROUP

All 4 cases of ethlene dibromide (EDB) expired with in 2 days of admission. In EDB poisoning patients presented with complaints of gastrointestinal upsets, after 4-6 hours of intake of drugs patients developed hypotension, tachycardia restlessness, oliguria, gastric bleeding, haemolysis, unconsciousness etc.

On investigation all 4 cases were positive electrocardiographic changes. They were atrial fibrillation. ST-T changes, ventricular ectopics and tachycardia. Blood urea, creatinine and serum enzyme levels SGOT, SGPT, CPKMB values raised in all 4 cases of poisoning whereas serum bilirubin raised only 3(75%) cases of EDB poisoning. Investigation results indicated that it is highly toxic substance and damaged to heart, liver and kidney etc.

Ethylene dibromide available in the market in the name of EDB ampoule, which contains 2 ml colourless fluid. It is used as a grain preservative.

OUTCOME OF TREATMENT

Even after giving proper treatment, 82(18.4%) cases were expired, in which mostly 56(68.3%) deaths were due to aluminium phosphide poisoning, and 12(14.6%) deaths due to organophosphorus poisoning cases.

Remaining 14(17.1%) deaths were due to rodenticide, alcohols, dhatura, EDB poisoning etc.

Almost similar type of results observed by Multani et al (1991) in which overall mortality in total poisoning cases was 25.5%. It was highest in aluminium phosphide poisoning 77.2% followed by mixed poisoning and organophosphorus poisoning.

PREVENTION OF POISONING

Accidental poisoning was common in children, it is prevented by :

1. Protection of child from poisonous substance, the poisonous substances should be kept secured places beyond the reach of the child.
2. Parents about the potential household poisons should be educated.
3. Need for parenteral supervision for toddler behaviour should be emphasized.
4. In proper technique of spraying of organophosphorus compounds (Insecticides) and proper way to put celphos tablets/EDB ampoules in stored grains with rubber gloves.
5. Stupefying poisoning prevented by never takes any substances such as biscuits, cigarettes, pans etc. from unknown persons or during journey.

6. Very toxic substances should be banned such as aluminium phosphide tablets.
7. All toxic substances (especially pesticides) should be provided with specific antidotes.
8. In the toxic substances regulation by state should be enforced.
9. Establishment of poison control centres to collect couple and disseminate information from poisons and their management.

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C O N C L U S I O N

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CONCLUSIONS

The following conclusionswere drawn from the present study :

1. Acute poisoning is widely prevalent in Bundelkhand region. Its incidence was 6/1000/year of total hospital admissions. Roughly 13th case of medical emergency was a poisoning case.
2. 71% cases belonged to rural and remaining 29% cases were from urban areas. Most of poisoning cases came from Uttar Pradesh (68.1%) and remaining 31.9% cases from Madhya Pradesh.
3. Around two third of total poisoning cases (67.4%) were between 15-34 years of age.
4. Around one fourth of cases of poisoning were less than 14 years of age (Pediatric age group) and most of them were of accidental poisoning.
5. The occurrence of acute poisoning was slightly more common in males (M : F = 1 : 0.85).
6. Poisoning were more common (85.2%) in Hindus and married people (53%).
7. Acute poisoning was more common in lower socio-economic status group (73.7%).
8. The intention of poisoning was suicidal in 75.5% cases, accidental in 19.8%, stupefying in 3.6% and homicidal in 1.1% cases.

9. Nearly half (45.7%) of poisoning cases admitted between months of April To July.
10. Aluminium phosphide poisoning was commonest in Bundelkhand region (20.5%) other commonest poisoning were dhatura, rodenticide and organophosphorus compounds.
11. Most of the cases (66.7%) of poisoning were discharged within 3 days.
12. 18.4% of poisoning cases were expired, 71% survived and remaining 10.5% cases were absconded.
13. Most of deaths of poisoning were due to aluminium phosphide (68.3%) and organophosphorus compounds (14.6%).
14. Most of death of poisoning cases occurred within 24 hours.
15. Mortality rate was higher in aluminium phosphide poisoning i.e. 61.5% followed by 19.7% by organophosphorus poisoning.
16. In aluminium phosphide poisoning cases mortality was higher in those patients who took fresh (62%) or more than 3 tablets (76.2 - 100%).
17. Most of cases of poisoning presented with gastrointestinal upsets. 45% of cases had positive electrocardiographic changes.
18. 9.2% of poisoning cases had psychiatric illness (depression and behaviour disorders).

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